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Case Report

Delayed onset sequential bilateral abducens nerve palsies secondary to traumatic CSF leak

Kavya Koshy [a,*], Marc Schnekenburger [b,c], Richard Stark [a], Mark Fitzgerald [b,c]

[a] Department of Neurology, The Alfred Hospital, Melbourne, Victoria, Australia
[b] Trauma Service, The Alfred Hospital, Melbourne, Victoria, Australia
[c] National Trauma Research Institute, Melbourne, Victoria, Australia

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ABSTRACT

Abducens nerve palsy via direct or indirect injury is well described following head trauma likely due to its long anatomical course with several vulnerable segments. However, bilateral abducens palsies due to non-iatrogenic intracranial hypotension is unique. This report describes the case of a male with sequential delayed onset abducens nerve palsies following head and neck trauma due to intracranial hypotension secondary to cerebrospinal fluid (CSF) leak from a dural tear at the C6/7 level. Signs of intracranial hypotension were evident on magnetic resonance imaging (MRI). We hypothesise that the traction effect from ongoing CSF leak resulted in sequential palsies. His clinical course was also complicated by pulmonary embolus and a prolonged period of immobility, the anti-gravity effects of which likely mitigated the CSF leak in the early period. Conservative management was undertaken with bed rest, fluids and caffeine with good response and resolving abducens dysfunction after ten weeks. Further management with epidural blood patch or surgical fixation was not necessary and deemed unlikely to succeed given the location of the dural tear and the need for concurrent anticoagulation. It is important to recognise CSF leak and intracranial hypotension as potential, albeit rare, causes for sequential abducens nerve palsy in patients with head and spinal injuries. Management strategies of this condition range from conservative measures to surgical intervention.

Introduction

Abducens palsy is an uncommon yet well described complication following head trauma due to either direct or indirect nerve injury along its tortuous intracranial course. Whilst indirect insult to the nerve is primarily attributed to ischaemia or vasospasm, direct mechanical injury is secondary to compression or avulsion of the nerve itself. Apart from the more common forms of compressive phenomena usually associated with head trauma such as skull fracture or haematoma, traction injury on the nerve from low CSF pressure after a CSF leak is also a potential cause of direct injury. Non-iatrogenic traumatic CSF leak is an extremely uncommon occurrence with most cases described in literature presenting as base of skull injuries. We describe a unique case of sequential bilateral abducens palsies following traumatic cervical spinal injury resulting in CSF leak. Apart from cranial neuropathies, CSF leak can cause severe headache, CSF fistula and central nervous system (CNS) infections; hence, it is important to recognise this phenomenon and monitor closely for complications.

* Corresponding author at: The Alfred Hospital, 55 Commercial Road, Melbourne, VIC 3004, Australia.
E-mail address: k.koshy@alfred.org.au (K. Koshy).

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Case presentation

A previously well 51-year-old male presented with injuries following a fall from a height of 4 m. He was initially assessed to have a Glasgow Coma Scale (GCS) of 15 and a normal cranial and peripheral nerve examination. He had sustained a 15-centimetre long occipital scalp laceration, bilateral C7 lamina fractures and a T1 superior vertebral body fracture (Fig. D). The spinal injuries were classified as Type B, C7/T1 (T1:A1) M1 as per the AO Spinal Classification.

An MRI scan demonstrated the full extent of the spinal injuries, including C7 posterior elements fractures, capsular injuries to bilateral C6–7 and C7-T1 facet joints, and tears of the ligamentum flavum and interspinous ligament at the level of C6–7. There was a small anterior and a large posterior epidural collection throughout the thoracic spine, with acute CSF leakage attributed to the dural tear at C6–7 level. The spinal injuries were managed non-operatively with a Miami-J collar.

The patient reported bilateral upper limb neuropathic pain that was treated with pregabalin. He also had mild fluctuating headaches and experienced some postural dizziness and was advised to restrict his mobility to avoid aggravating the CSF leak.

The patient developed extensive bilateral pulmonary emboli on day four with confirmed bilateral lower limb deep vein thromboses on Doppler ultrasound. Anticoagulation had to be suspended on day nine due to a spontaneous quadriceps haematoma, further limiting his mobility. An IVC filter was inserted and after a three-day hiatus, therapeutic anticoagulation was recommenced without further bleeding complications. Following this, the patient was able to engage with light physiotherapy.

On day thirteen, the patient reported acute onset binocular horizontal diplopia worse on right gaze with clinical features in keeping with a partial right abducens nerve palsy (Fig. A). This was managed conservatively with an eye patch. Two days later, the patient was noted to have a left-sided abducens palsy (Fig. B) with some improvement in the right-sided dysfunction. There were no other neurological deficits. MRI and computed tomography (CT) with venography of the brain did not demonstrate thrombus, stroke, demyelination or direct injury to the abducens nerve. The imaging did however demonstrate the presence of hygromas and mild cerebellar tonsillar herniation suggestive of intracranial hypotension. These results coupled with the known dural tear at the C6/7 level (Fig. C) confirmed the diagnosis.

Diagnosis

The patient was diagnosed with sequential bilateral abducens palsies secondary to a traumatic CSF leak and associated intracranial hypotension. The late onset of his symptoms was attributed to the prolonged period of bed rest prescribed immediately after the trauma, the anti-gravity effects of which may have mitigated the CSF leak initially. Following consultation with the radiology, anaesthetics and spinal teams, the case was deemed too high risk for epidural blood patch or surgical repair of the dural tear, given the high spinal location, technical difficulty due to size and location of the leak and the need for ongoing therapeutic anticoagulation to prevent further clot progression. The patient was discharged home and monitored in the neuro-ophthalmology clinic where he was observed to have full resolution of the right abducens nerve palsy and mild left abducens nerve palsy after ten weeks.

Differential diagnosis

The aetiology of bilateral abducens nerve palsy includes cerebrovascular disease, intracranial hypertension or hypotension, carotid-cavernous fistulas, infection, trauma, demyelination and tumours. Traumatic aetiologies are primarily attributed to skull fractures, haematoma and extremes of CSF pressure all of which can result in injury to the nerve [1]. In our case, there was no radiologically discernible injury to the nerves from the trauma nor any signs of intracranial hypertension; however, there was evidence of low CSF pressure presumed due to leaking CSF, associated over time with sequential bilateral abducens nerve dysfunction. Abducens nerve palsy due to intracranial hypotension (ICH) is a recognised phenomenon but has only been previously described in cases of iatrogenic or spontaneous ICH [2,3]. The oculomotor and trochlear nerves can also be affected by ICH [2,3].

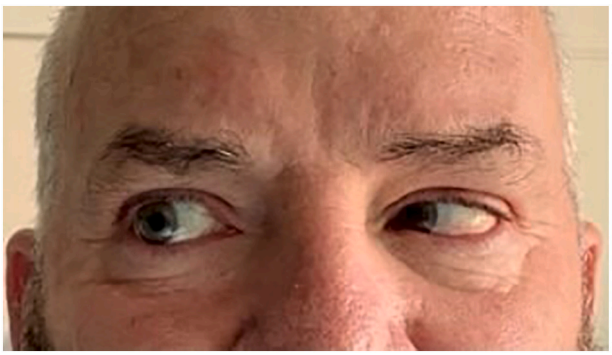

Fig. A. Mild right-sided abduction deficit on right gaze.

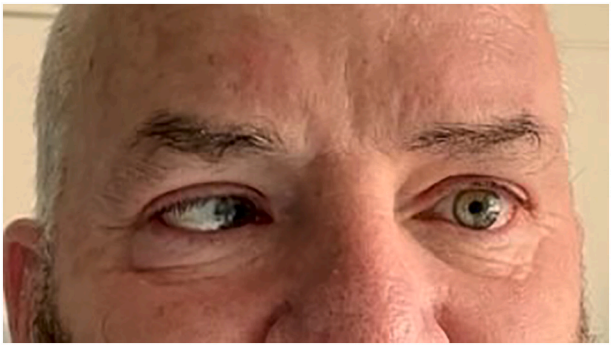

Fig. B. Severe left-sided abduction deficit on left gaze.

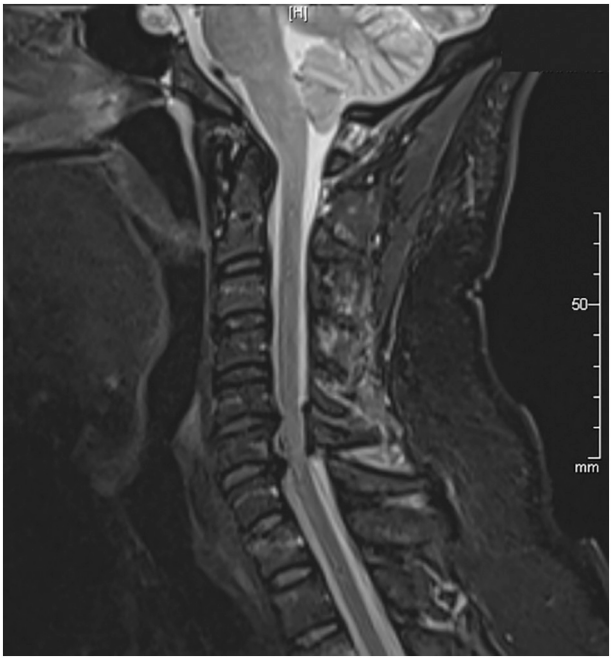

Fig. C. Sagittal T2-STIR MRI of cervical spine demonstrating disrupted ligamentum flavum at C5-6 and C6-7 with corresponding posterior intraspinal CSF collection with partial effacement and cerebellar tonsillar inferior descent.

Discussion

Understanding the anatomical course of the abducens nerve is essential in appreciating the various aetiologies of its dysfunction. The nerve arises from the dorsal pons which receives its vascular supply from the basilar artery. The nerve fibres leave the brainstem through the pontomedullary groove in close proximity to the facial and vestibulocochlear nerves before travelling through the subarachnoid space. The abducens nerve then crosses the temporal bone toward the clivus anchored within Dorello's canal, a fibrous sheath demarcated by Gruber ligament, the posterior clinoidal process and the petrous bone. This particular segment of the nerve leaves it vulnerable to compression from fractures, compression or fluctuations in CSF pressure. Finally, it courses through the cavernous sinus in close proximity to the internal carotid artery, before reaching the lateral rectus muscle through the superior orbital fissure.

In our case, we hypothesise that the gradual progression of CSF leak from the C6/7 dural tear resulted in intracranial hypotension and worsening traction on the abducens nerve within Dorello's canal. Whilst it is unusual that the patient did not report substantial headache, it should be noted that this may have been confounded by the analgesic regimen prescribed for his traumatic injuries.

Treatment

The treatment for delayed abducens neve palsy secondary to trauma is almost always non-operative. For uncomplicated cases,

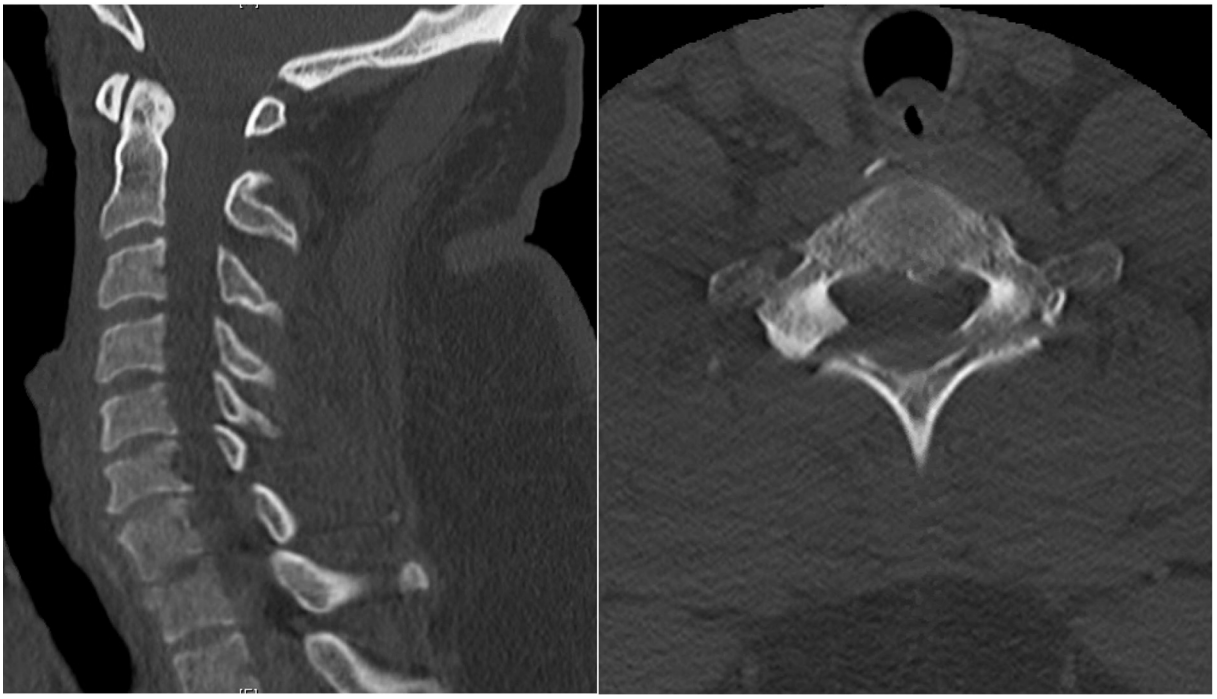

Fig. D. CT image of cervical spine demonstrating minimally displaced C7 bilateral lamina fractures extending into C7-T1 facet joints and anterior cortical step involving T1 with approximately 20% loss of height.

conservative measures such as bed rest, hydration and generous caffeine intake are recommended, with emphasis on symptomatic management of diplopia by using eye patching or prism lenses. If there are complications such as severe deficits, dizziness or headache, an epidural blood patch (EBP) may be effective [4]. There is some evidence for surgical repair of the dura for cases that are refractory to conservative management or EBP. However, the benefits are limited to cases in which a clear site of CSF leak has been identified [5].

Conclusion

In cases of traumatic bilateral abducens nerve palsies, particularly with a delayed onset and sequential progression, a diagnosis of intracranial hypotension due to CSF leak should be considered with close attention for potential complications. MR and CT imaging are useful to exclude other causes such as ischaemia, direct nerve injury, demyelination, fracture or thrombosis, and may reveal signs of underlying intracranial hypotension.

Declaration of competing interest

The authors of this paper declare they have no relevant conflicts of interest.

CRediT authorship contribution statement

Kavya Koshy: Conceptualisation, Writing – Original draft, Writing – Review and editing.

Marc Schnekenburger: Conceptualisation, Writing – Original draft, Writing – Review and editing.

Richard Stark: Writing – Review and editing.

Mark Fitzgerald: Writing – Review and editing.

The patient gave consent for publication, as outlined by the Alfred Hospital Ethics and Review Board. The authors acknowledge the support of the Trauma, Neurology and Radiology Service units in the formulation of this manuscript.

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